Amendments to the Specification:

Please amend the specification as follows:

Please replace the paragraph starting at page 4, line 13, with the following rewritten paragraph:

These datea support *Math1* as an essential factor in the control of HC differentiation. To determine whether expression of *Math1* was required for the roscovitine-induced appearance of supernumeracy HC, the inventors uses of cultured organs of Corti from the *Math1*$^{-}$ mice. In *Math1*-null mice, the treatment of E`5.5 organs of Corti with roscovitine does not induce the appearance of HCs after 5DIV. However, in heterozygote mice, ~~⊟-gal~~ beta-gal positive super-numeracy HCs arose. Taken together, these results demonstrate that the induction of HCs by roscovitine likely recapitulates the developmental pattern of HC development which is drastically Math1 –dependent.

Please replace the paragraph bridging pages 6 and 7, with the following rewritten paragraph:

FIG. 4: Roscovitine effects: dose-response (A), kinetics (B) and developmental stage specificity (C): (A) dose-response curve for roscovitine-induced supernumerary HCs in E19 cultured organ of Corti explants. Each data bar represents the mean length of supernumerary HCs regions for a minum of 4 explants per experiment. (B) The mean length of supernumerary HCs regions in control condition or in the presence of roscovitine (10 :M) was calculated as a function of time in culture with E19 rat organ of Corti explants. (C) Developmental stage dependence of roscovitine-induced supernumerary cells. Ten :M roscovitine was added to rat organ of Corti explants dissected from stages B17, E19, P0, P2 and P4. The mean length of supernumerary HCs regions was monitored after 5 days of culture. Results were expressed as mean ~~∀-⊟~~ ∀ ± sem (n=5). Statistical significance was determined using a Student's t-test *=p<0.05 and ***=p<0.001.

Please replace the paragraph beginning on page 13, line 24, with the following rewritten paragraph:

p9^{CKShs1}-sepharose affinity purification was also used to determine the histone H1 kitase activity of bound CDK1 and CDK2. After purification as described above, the p9.sup.CKShs1-sepharose kinases were incubated for 30 min at 30° C. with 1 ~~⊟Ci~~ microCi [^{32}P] ATP (1-3 Ci/mmol, Amersham) in the presence of 25 µg histone H1 (Type III-S, Sigma) in a final volume of 30 µl of buffer C (homogenization buffer but 5 mM EGTA, no NaF and no protease inhibitors). Assays were terminated by transferring the tube on ice. 30 :l 2X Laenmli sample buffer was added. Phosphorylation of the substrate was assessed by autoradiography after SDS-PAGE.